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SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: Dwayne C. Jones Examiner # : 71299 Date: 15 JAN 02
Art Unit: 1614 Phone Number 308-4634 Serial Number: 09/787,491
Mail Box and ~~Bldg~~/Room Location: 2007, CM1 Results Format Preferred (circle): PAPER DISK E-MAIL
2001, CM1

If more than one search is submitted, please prioritize searches in order of need.

****

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: see attached sheet

Inventors (please provide full names): 11

Earliest Priority Filing Date: 11

***For Sequence Searches Only* Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.**

Please search claims 1-3

POINT OF CONTACT:
EARB O'BRYEN
TECH. INFORMATION SPECIALIST
STIC CM1 ~~12B14~~ 308-4291
12E18

****

STAFF USE ONLY	Type of Search	Vendors and cost where applicable
Searcher: BOB	NA Sequence (#) _____	STN 176
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Calculated physical property data is now available.  See HELP PROPERTIES
for more information.  See STNote 27, Searching Properties in the CAS
Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf



L3   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2002 ACS
RN   27750-10-3  REGISTRY
CN   D-erythro-Pentaric acid, 3-C-carboxy-2-deoxy- (8CI, 9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   (-)-2-Hydroxycitric acid
CN   (-)-Hydroxycitric acid
CN   Citric acid, 2-hydroxy-, (-)-
CN   Garcinia acid
CN   Hydroxycitric acid
FS   STEREOSEARCH
DR   4373-35-7
MF   C6 H8 O8
CI   COM
LC   STN Files:   ADISNEWS, AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS,
       BIOTECHNO, CA, CAOLD, CAPLUS, CIN, DDFU, DRUGU, EMBASE, HODOC*, IPA,
       NAPRALERT, NIOSHTIC, PROMT, TOXCENTER, TOXLIT, USPATFULL
         (*File contains numerically searchable property data)

Absolute stereochemistry.
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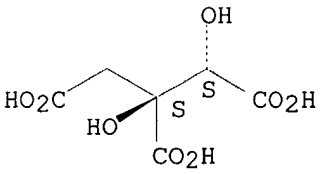


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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

           102 REFERENCES IN FILE CA (1967 TO DATE)
             4 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
           102 REFERENCES IN FILE CAPLUS (1967 TO DATE)
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=> fil capl; d que l13; fil medl; d que l22; fil biosis; d que l31; d que l33; d que l34; s l33 or l34
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FILE LAST UPDATED: 16 Jan 2002 (20020116/ED)

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L2	5	SEA FILE=REGISTRY ABB=ON 27750-10-3 OR 64913-19-5 OR 132436-67-0 OR 185196-38-7 OR 213385-58-1
L4	162	SEA FILE=CAPLUS ABB=ON L2 OR (HYDROXYCITRIC OR HYDROXY CITRIC OR GARCINIA)(W)ACID
L5	1	SEA FILE=REGISTRY ABB=ON INSULIN/CN
L6	70022	SEA FILE=CAPLUS ABB=ON L5
L7	139785	SEA FILE=CAPLUS ABB=ON ?INSULIN?
L8	30876	SEA FILE=CAPLUS ABB=ON HYPERTENSION/CT
L9	22539	SEA FILE=CAPLUS ABB=ON BLOOD PRESSURE/CT
L10	16906	SEA FILE=CAPLUS ABB=ON GLUCOCORTICOIDS+NT,OLD/CT
L11	20335	SEA FILE=CAPLUS ABB=ON ANTIHYPERTENSIVES/CT
L13	13	SEA FILE=CAPLUS ABB=ON L4 AND (L6 OR L7 OR L8 OR L9 OR L10 OR L11)

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FILE LAST UPDATED: 2 JAN 2002 (20020102/UP). FILE COVERS 1958 TO DATE.

On April 22, 2001, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE now contains IN-PROCESS records. See HELP CONTENT for details.

MEDLINE is now updated 4 times per week. A new current-awareness alert frequency (EVERYUPDATE) is available. See HELP UPDATE for more information.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2001 vocabulary. Enter HELP THESAURUS for details.

The OLDMEDLINE file segment now contains data from 1958 through 1965. Enter HELP CONTENT for details.

Left, right, and simultaneous left and right truncation are available in the Basic Index. See HELP SFIELDS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

```
L15          124 SEA FILE=MEDLINE ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR
                 GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC)(W)ACID
L16        23929 SEA FILE=MEDLINE ABB=ON  ANTIHYPERTENSIVE AGENTS/CT
L17       149166 SEA FILE=MEDLINE ABB=ON  HYPERTENSION+NT/CT
L18       162221 SEA FILE=MEDLINE ABB=ON  BLOOD PRESSURE+NT/CT
L19         1076 SEA FILE=MEDLINE ABB=ON  HYPERINSULINEMIA/CT
L20        97127 SEA FILE=MEDLINE ABB=ON  INSULIN+NT/CT
L21        92548 SEA FILE=MEDLINE ABB=ON  GLUCOCORTICOIDS+NT/CT
L22           16 SEA FILE=MEDLINE ABB=ON  L15 AND (L16 OR L17 OR L18 OR L19 OR
                 L20 OR L21)
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 16 January 2002 (20020116/ED)

The BIOSIS file has been reloaded.  Enter HELP RLOAD and HELP REINDEXING
for details.
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L2             5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                 -0 OR 185196-38-7 OR 213385-58-1
L15          124 SEA FILE=MEDLINE ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR
                 GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC)(W)ACID
L25          132 SEA FILE=BIOSIS ABB=ON  L2 OR L15
L26       189636 SEA FILE=BIOSIS ABB=ON  ?HYPERTENS?
L27       113404 SEA FILE=BIOSIS ABB=ON  BLOOD PRESSURE
L31            0 SEA FILE=BIOSIS ABB=ON  L25 AND (L26 OR L27)
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L2             5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                 -0 OR 185196-38-7 OR 213385-58-1
L15          124 SEA FILE=MEDLINE ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR
                 GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC)(W)ACID
L25          132 SEA FILE=BIOSIS ABB=ON  L2 OR L15
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L29       31415 SEA FILE=BIOSIS ABB=ON  GLUCOCORTICOID# OR HYDROXYCORTICOSTEROI
                D#
L33           1 SEA FILE=BIOSIS ABB=ON  L25 AND L29


L2            5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                -0 OR 185196-38-7 OR 213385-58-1
L15         124 SEA FILE=MEDLINE ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR
                GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC)(W)ACID
L25         132 SEA FILE=BIOSIS ABB=ON  L2 OR L15
L28      194934 SEA FILE=BIOSIS ABB=ON  ?INSULIN?
L34           5 SEA FILE=BIOSIS ABB=ON  L25(IOA)L28


L77           6 L33 OR L34

=> fil biotechno; d que l39; fil drugu; d que l45; fil embase; d que l54; fil ipa; d que
l56; fil napra; d que l62; fil wpids;d  que l68
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>>>  SIMULTANEOUS LEFT AND RIGHT TRUNCATION AVAILABLE IN
     /CT AND BASIC INDEX  <<<


L2            5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                -0 OR 185196-38-7 OR 213385-58-1
L15         124 SEA FILE=MEDLINE ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR
                GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC)(W)ACID
L35          19 SEA FILE=BIOTECHNO ABB=ON  L2 OR L15
L36       11321 SEA FILE=BIOTECHNO ABB=ON  ?HYPERTENS? OR BLOOD PRESSURE
L37       33077 SEA FILE=BIOTECHNO ABB=ON  ?INSULIN?
L38        9067 SEA FILE=BIOTECHNO ABB=ON  GLUCOCORTICOID# OR HYDROXYCORTICOSTE
                ROID#
L39           4 SEA FILE=BIOTECHNO ABB=ON  L35 AND (L36 OR L37 OR L38)


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>>>  SEE HELP COST                                       <<<

>>>  FILE COVERS 1983 TO DATE  <<<
>>>  THESAURUS AVAILABLE IN /CT  <<<


L2            5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                -0 OR 185196-38-7 OR 213385-58-1
L15         124 SEA FILE=MEDLINE ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR
                GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC)(W)ACID
L40          24 SEA FILE=DRUGU ABB=ON  L2 OR L15
```

```
L41        54284 SEA FILE=DRUGU ABB=ON  ANTIHYPERTENS? OR HYPERTENS?
L42        57758 SEA FILE=DRUGU ABB=ON  BLOOD PRESSURE
L43        27225 SEA FILE=DRUGU ABB=ON  HYPERINSULIN? OR INSULIN
L44         6232 SEA FILE=DRUGU ABB=ON  GLUCOCORTICOID# OR HYDROXYCORTICOSTEROID
                 # OR (GLUCO CORTICOID#) OR (HYDROXYCORTICO OR HYDROXY CORTICO)(
                 W)STEROID#
L45            6 SEA FILE=DRUGU ABB=ON  L40 AND (L41 OR L42 OR L43 OR L44)

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 FILE COVERS 1974 TO 17 Jan 2002 (20020117/ED)

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L46          106 SEA FILE=EMBASE ABB=ON  HYDROXYCITRIC ACID/CT
L47        16808 SEA FILE=EMBASE ABB=ON  ANTIHYPERTENSIVE AGENT/CT
L48         2958 SEA FILE=EMBASE ABB=ON  ANTIHYPERTENSIVE ACTIVITY/CT
L49       153770 SEA FILE=EMBASE ABB=ON  HYPERTENSION+NT/CT
L50       127532 SEA FILE=EMBASE ABB=ON  BLOOD PRESSURE+NT/CT
L51         5737 SEA FILE=EMBASE ABB=ON  HYPERINSULINEMIA/CT
L52        85220 SEA FILE=EMBASE ABB=ON  INSULIN/CT
L53       208169 SEA FILE=EMBASE ABB=ON  GLUCOCORTICOID+NT/CT
L54           15 SEA FILE=EMBASE ABB=ON  L46 AND (L47 OR L48 OR L49 OR L50 OR
                 L51 OR L52 OR L53)

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FILE COVERS 1970 TO 3 JAN 2002 (20020103/ED)

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L2             5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                 -0 OR 185196-38-7 OR 213385-58-1
L56            2 SEA FILE=IPA ABB=ON  L2 OR HYDROXYCITRATE OR (HIBISCUS OR
                 GARCINIA OR HYDROXYCITRIC OR HYDROXY-CITRIC OR HYDROCITRIC)(W)A
                 CID

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MOST RECENT DERWENT UPDATE            200204   <200204/DW>
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http://www.derwent.com/chemistryresource/index.html <<<

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SEE http://www.derwent.com/dwpi/updates/dwpicov/index.html <<<

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L63          43 SEA FILE=WPIDS ABB=ON  HYDROXYCITRATE OR (HIBISCUS OR GARCINIA
                OR HYDROXYCITRIC OR HYDROXY CITRIC OR HYDROCITRIC)(W)ACID
L64       15898 SEA FILE=WPIDS ABB=ON  ?HYPERTENS?
L65        6894 SEA FILE=WPIDS ABB=ON  BLOOD PRESSURE
L66        8124 SEA FILE=WPIDS ABB=ON  ?INSULIN?
L67        1100 SEA FILE=WPIDS ABB=ON  GLUCOCORTICOID# OR HYDROXYCORTICOSTEROID
                # OR (GLUCO CORTICOID#) OR (HYDROXYCORTICO OR HYDROXY CORTICO)(
                W)STEROID#
L68           2 SEA FILE=WPIDS ABB=ON  L63 AND (L64 OR L65 OR L66 OR L67)

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L2            5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                -0 OR 185196-38-7 OR 213385-58-1
L70         205 SEA HYDROXYCITRATE OR (HIBISCUS OR GARCINIA OR HYDROXYCITRIC
                OR HYDROXY CITRIC OR HYDROCITRIC)(W) ACID
L71           5 SEA L2
L72      400494 SEA HYPERTENS? OR ANTIHYPERTENS? OR BLOOD PRESSURE
L73           1 SEA (L70 OR L71) AND L72

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L2            5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                -0 OR 185196-38-7 OR 213385-58-1
L74          91 SEA HYDROXYCITRATE OR (HIBISCUS OR GARCINIA OR HYDROXYCITRIC
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```
                OR HYDROXY CITRIC OR HYDROCITRIC)(W) ACID OR L2
L75         39224 SEA HYPERTENS? OR ANTIHYPERTENS? OR BLOOD PRESSURE
L76             7 SEA L74(P) L75

=> dup rem l22,l56,l13,l77,l39,l54,l45,l68,l73,l76
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PROCESSING COMPLETED FOR L45
PROCESSING COMPLETED FOR L68
PROCESSING COMPLETED FOR L73
PROCESSING COMPLETED FOR L76
L78            47 DUP REM L22 L56 L13 L77 L39 L54 L45 L68 L73 L76 (25 DUPLICATES REMOVED)
                ANSWERS '1-16' FROM FILE MEDLINE
                ANSWERS '17-18' FROM FILE IPA
                ANSWERS '19-25' FROM FILE CAPLUS
                ANSWER '26' FROM FILE BIOSIS
                ANSWERS '27-33' FROM FILE EMBASE
                ANSWERS '34-38' FROM FILE DRUGU
                ANSWER '39' FROM FILE WPIDS
                ANSWER '40' FROM FILE ADISALERTS
                ANSWERS '41-42' FROM FILE USPATFULL
                ANSWERS '43-47' FROM FILE EUROPATFULL

=> d ibib ab hitrn l78 1-47
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L78 ANSWER 1 OF 47 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000184967 MEDLINE
DOCUMENT NUMBER: 20184967 PubMed ID: 10721892
TITLE: The role of long-chain fatty acyl-CoA esters in beta-cell signal transduction.
AUTHOR: Corkey B E; Deeney J T; Yaney G C; Tornheim K; Prentki M
CORPORATE SOURCE: Department of Medicine, Boston University Medical School, MA 02118, USA.
CONTRACT NUMBER: DK 46200 (NIDDK)
DK35914 (NIDDK)
DK50662 (NIDDK)
SOURCE: JOURNAL OF NUTRITION, (2000 Feb) 130 (2S Suppl) 299S-304S.
Ref: 52
Journal code: JEV; 0404243. ISSN: 0022-3166.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000421
Last Updated on STN: 20000421
Entered Medline: 20000412

AB Glucose-induced insulin secretion is associated with inhibition of free fatty acid (FFA) oxidation, increased esterification and complex lipid formation by pancreatic beta-cells. Abundant evidence favors a role for cytosolic long-chain acyl-CoA (LC-CoA), including the rapid rise in malonyl CoA, the inhibitory effect of **hydroxycitrate** or acetyl CoA carboxylase knockout, both of which prevent malonyl CoA formation, and the stimulatory effect of exogenous FFA. On the other hand, some evidence opposes the concept, including the fall in total LC-CoA levels in response to glucose, the stimulatory effect of LC-CoA on K(ATP) channels and the lack of inhibition of glucose-stimulated secretion either by overexpression of malonyl CoA decarboxylase, which markedly lowers malonyl CoA levels, or by triacsin C, which blocks FFA conversion to LC-CoA. Alternative explanations for these data are presented. A revised model of nutrient-stimulated secretion involving two arms of signal transduction that occur simultaneously is proposed. One arm depends on modulation of the K(ATP) channel evoked by changes in the ATP/ADP ratio. The other arm depends upon anaplerotic input into the tricarboxylic acid cycle, generation of excess citrate, and increases in cytosolic malonyl-CoA. Input from this arm is increased LC-CoA. Signaling through both arms would be required for normal secretion. LC-CoA esters and products formed from them are potent regulators of enzymes and channels. It is hypothesized that their elevations directly modulate the activity of enzymes, genes and various beta-cell functions or modify the acylation state of key proteins involved in regulation of ion channels and exocytosis.

L78 ANSWER 2 OF 47 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97344123 MEDLINE
DOCUMENT NUMBER: 97344123 PubMed ID: 9200650
TITLE: Stimulation of islet protein kinase C translocation by palmitate requires metabolism of the fatty acid.
AUTHOR: Alcazar O; Qiu-yue Z; Gine E; Tamarit-Rodriguez J
CORPORATE SOURCE: Department of Biochemistry, Complutense University Medical School, Madrid, Spain.
SOURCE: DIABETES, (1997 Jul) 46 (7) 1153-8.
Journal code: E8X; 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970724
Last Updated on STN: 19970724
Entered Medline: 19970716

AB The secretory, metabolic, and signaling aspects of glucose/palmitate interaction on beta-cell function have been studied on rat islets. Palmitate potentiated the glucose-induced insulin response of perifused islets at suprathreshold (>3 mmol/l) sugar concentrations. This potentiating effect could be suppressed by 8-bromo-cGMP, which also blocks palmitate metabolism. Palmitate did not modify glucose utilization, but it slightly reduced glucose oxidation and concomitantly increased lactate production. The very low rate of palmitate oxidation (80-fold lower than that of 20 mmol/l glucose) might explain its lack of effect on glycolysis and hence that the glucose/fatty acid cycle is inoperative in islet cells. However, glucose determines the metabolic fate of exogenous palmitate, which is mainly diverted toward lipid synthesis at high sugar concentrations and might then generate lipid messengers for cell signaling. Palmitate did not increase glucose-induced production of inositol-1,4,5-trisphosphate, but it stimulated the translocation of protein kinase C activity from a cytosolic to a particulate fraction at 20 but not at 3 mmol/l glucose. This increased translocation was partially or completely blocked by **hydroxycitrate** or 8-bromo-cGMP, respectively, which are agents interfering with palmitate metabolism (inhibiting lipid synthesis). The metabolic interaction between glucose and palmitate might generate lipid messengers (diacylglycerol, phosphatidylserine) necessary for the activation of islet protein kinase C, which would in turn result in a potentiation of glucose-induced insulin secretion.

L78 ANSWER 3 OF 47 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 92144681 MEDLINE
DOCUMENT NUMBER: 92144681 PubMed ID: 1782221
TITLE: Hexose metabolism in pancreatic islets. Effect of (-)-**hydroxycitrate** upon fatty acid synthesis and insulin release in glucose-stimulated islets.
AUTHOR: Sener A; Malaisse W J
CORPORATE SOURCE: Laboratory of Experimental Medicine, Brussels Free University, Belgium.
SOURCE: BIOCHIMIE, (1991 Oct) 73 (10) 1287-90.
Journal code: A14; 1264604. ISSN: 0300-9084.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199203
ENTRY DATE: Entered STN: 19920405
Last Updated on STN: 19920405
Entered Medline: 19920313

AB Anaplerotic reactions leading to the de novo synthesis of fatty acids were recently proposed to participate in the coupling of metabolic to secretory events in the process of glucose-stimulated insulin release. In an attempt to validate such a proposal, the effect of (-)-**hydroxycitrate** upon fatty acid synthesis and insulin release was investigated in glucose-stimulated rat pancreatic islets. The inhibitor of ATP citrate-lyase, when tested in the 1.0-2.0 mM range, failed to affect glucose-stimulated insulin release, but also failed to inhibit the incorporation of 14C-labelled acetyl residues derived from L-[U-14C]leucine into islet lipids. A partial inhibition of fatty acid labelling by 3H2O was only observed in islets incubated for 120 min in the presence of 5.0 mM (-)-**hydroxycitrate** and absence of CaCl2. These findings suggest that (-)-**hydroxycitrate** is not, under the present experimental conditions, a useful tool to abolish fatty acid synthesis in intact pancreatic islets.

L78 ANSWER 4 OF 47 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 90254190 MEDLINE
DOCUMENT NUMBER: 90254190 PubMed ID: 2160286
TITLE: Glucocorticoid induction of fatty-acid synthase mediates the stimulatory effect of the hormone on choline-phosphate cytidylyltransferase activity in fetal rat lung.
AUTHOR: Xu Z X; Smart D A; Rooney S A
CORPORATE SOURCE: Department of Pediatrics, Yale University School of Medicine, New Haven, CT.
CONTRACT NUMBER: HD-10192 (NICHD)
HL-43320 (NHLBI)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1990 May 1) 1044 (1) 70-6.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199006
ENTRY DATE: Entered STN: 19900720
Last Updated on STN: 19980206
Entered Medline: 19900628

AB Fetal lung fatty-acid synthase and choline-phosphate cytidylyltransferase activities are increased by glucocorticoids. There is evidence that the hormone increases synthesis of fatty-acid synthase but only increases the catalytic activity of the cytidylyltransferase. Free fatty acids and a number of phospholipids have been reported to stimulate cytidylyltransferase activity in several organs, including the lung. We have addressed the question of whether glucocorticoid induction of fatty-acid synthase mediates the stimulatory effect of the hormone on choline-phosphate cytidylyltransferase activity. Explants of 18-day fetal rat lung were cultured for 48 h with dexamethasone and inhibitors of de novo fatty acid biosynthesis (agaric acid and **hydroxycitric acid**) being included in the medium for the final 20 h. Dexamethasone increased the activities of fatty acid synthase and choline-phosphate cytidylyltransferase by 84% and 60%, respectively. Agaric acid and **hydroxycitric acid** completely abolished the stimulatory effect of the hormone on cytidylyltransferase but not on fatty-acid synthase. The inhibitors had no effect on cytidylyltransferase activity in control cultures. Fetal lung choline-phosphate cytidylyltransferase can be maximally stimulated by inclusion of phosphatidylglycerol in the assay mixture and under this condition, cytidylyltransferase activity in control and dexamethasone-treated cultures in the presence and absence of the inhibitors were all increased to the same level. Therefore, the inhibitors did not diminish the capacity of cytidylyltransferase to be fully activated. We suggest that the glucocorticoid induction of fatty-acid synthase in fetal lung results in increased synthesis of fatty acids which in turn, either as free acids or after incorporation into phospholipids, activate choline-phosphate cytidylyltransferase.

L78 ANSWER 5 OF 47 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 85258111 MEDLINE
DOCUMENT NUMBER: 85258111 PubMed ID: 3894050
TITLE: Effect of drugs, peptide hormones and lipogenic precursors on the relative incorporation of [3H]H2O and carbon into hepatic cholesterol.
AUTHOR: Bjornsson O G; Pullinger C R; Gibbons G F
SOURCE: FEBS LETTERS, (1985 Aug 5) 187 (2) 302-6.
Journal code: EUH; 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198509
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19850924

AB Measurement of the weight of desmosterol produced during its biosynthesis in the presence of tritiated water and triparanol has permitted a direct determination of the relative flux of carbon and tritium (the H/C ratio) into sterol in hepatocytes. The H/C ratio increased with time of incubation irrespective of the nutritional state of the donor animals. This increase was more marked in hepatocytes from starved animals. Pyruvate and lactate increased, and glucagon decreased, the sterol H/C ratio. Addition of pyruvate to incubations containing glucagon resulted in a 32-67% increase in the H/C ratio depending upon nutritional status. Insulin had no effect whilst (-)-**hydroxycitrate** decreased the ratio by 25%.

L78 ANSWER 6 OF 47 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 83256412 MEDLINE
DOCUMENT NUMBER: 83256412 PubMed ID: 6135416
TITLE: Interactions between insulin and thyroid hormone in the control of lipogenesis.
AUTHOR: Sugden M C; Steare S E; Watts D I; Palmer T N
SOURCE: BIOCHEMICAL JOURNAL, (1983 Mar 15) 210 (3) 937-44.
Journal code: 9YO; 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198308
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19950206
Entered Medline: 19830811

AB 1. The effects of intragastric glucose feeding and L-tri-iodothyronine (T3) administration on rates of hepatic and brown-fat lipogenesis in vivo were examined in fed and 48 h-starved rats. 2. T3 treatment increased hepatic lipogenesis in the fed but not the starved animals. Brown-fat lipogenesis was unaffected or slightly decreased by T3 treatment of fed or starved rats. 3. Intragastric glucose feeding increased hepatic lipogenesis in control or T3-treated fed rats, but did not increase hepatic lipogenesis in starved control rats. Glucose feeding increased hepatic lipogenesis if the starved rats were treated with T3. Glucose feeding increased rates of brown-fat lipogenesis in all experimental groups. The effects of glucose feeding on liver and brown-fat lipogenesis were mimicked by insulin injection. 4. The increase in hepatic lipogenesis in T3-treated 48 h-starved rats after intragastric glucose feeding was prevented by short-term insulin deficiency, but not by (-)-**hydroxycitrate**, an inhibitor of ATP citrate lyase. The increase in lipogenesis in brown adipose tissue in response to glucose feeding was inhibited by both short-term insulin deficiency and (-)-**hydroxycitrate**. 5. The results tend to preclude pyruvate kinase and acetyl-CoA carboxylase as the sites of interaction of insulin and T3 in the regulation of hepatic lipogenesis in 48 h-starved rats. Other potential sites of interaction are discussed.

L78 ANSWER 7 OF 47 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 83027231 MEDLINE
DOCUMENT NUMBER: 83027231 PubMed ID: 6751811
TITLE: Effects of insulin and glucagon on fatty acid synthesis from acetate by hepatocytes incubated with (--)-**hydroxycitrate**.
AUTHOR: Beynen A C; Geelen M J
SOURCE: ENDOKRINOLOGIE, (1982 Jun) 79 (2) 308-10.

Journal code: EHJ; 0370675. ISSN: 0013-7251.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198212
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19821216

AB (--)-**Hydroxycitrate** is a well-known inhibitor of the citrate cleavage enzyme (EC 4.1.3.8). In isolated hepatocytes it inhibits fatty acid synthesis from glucose, but it does not affect fatty acid synthesis from acetate. In its presence, insulin stimulates and glucagon inhibits incorporation of labelled acetate into fatty acids. This is evidence that both hormones directly influence fatty acid synthesis from acetate.

L78 ANSWER 8 OF 47 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 82232390 MEDLINE
DOCUMENT NUMBER: 82232390 PubMed ID: 7046828
TITLE: Brown-adipose-tissue lipogenesis in starvation: effects of insulin and (-) **hydroxycitrate.**
AUTHOR: Sugden M C; Watts D I; Marshall C E; McCormack J G
SOURCE: BIOSCIENCE REPORTS, (1982 May) 2 (5) 289-97.
Journal code: A6D; 8102797. ISSN: 0144-8463.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198209
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19970203
Entered Medline: 19820924

AB Glucose or insulin increased lipogenesis (measured in vivo using 3H2O) in brown fat of starved rats. Such increases were associated with activation of pyruvate dehydrogenase and increased use of glucose as a lipogenic precursor (monitored as an increase in the 14C/3H ratio in brown-fat fatty acids in rats injected with both 3H20 and [U-14C]glucose). (-) **Hydroxycitrate** did not inhibit basal rates of brown-fat lipogenesis in starved rats but suppressed the increases in lipogenesis and glucose utilization observed in response to insulin. (-) **Hydroxycitrate** did not, however, inhabit the increase in 14C/3H observed after insulin treatment. The results indicate that in brown fat, glucose is utilized for fatty-acid synthesis predominantly via citrate, and that insulin acts to increase lipogenesis at site(s) prior to citrate cleavage. As basal rates of lipogenesis were not inhibited by (-) **hydroxycitrate,** it is suggested that acetate may be a lipogenic substrate for brown fat in starvation, and experiments are described which support this suggestion.

L78 ANSWER 9 OF 47 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 82068175 MEDLINE
DOCUMENT NUMBER: 82068175 PubMed ID: 7030319
TITLE: Effects of lactation on L-leucine metabolism in the rat. Studies in vivo and in vitro.
AUTHOR: Vina J R; Williamson D H
CONTRACT NUMBER: AM-11748 (NIADDK)
SOURCE: BIOCHEMICAL JOURNAL, (1981 Mar 15) 194 (3) 941-7.
Journal code: 9YO; 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198201

ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19820109

AB 1. The turnover rate of L-[1-14C]leucine was increased by 35% in lactating rats compared with virgin rats. Starvation or removal of pups (24 h) returned the value to that of the virgin rat. 2. Incorporation of L-[U-14C]leucine into lipid and protein of mammary glands of lactating rats in vivo increased 7-fold and 6-fold respectively compared with glands of virgin rats. Lactation caused no change in the incorporation of L-[U-14C]leucine into hepatic lipid and protein. 3. The production of 14CO2 from L[1-14C]leucine (in the presence of glucose) was similar in isolated acini from glands of fed (chow) and starved lactating rats. Feeding with a 'cafeteria' diet caused a slight decrease, and removal of pups a large decrease, in the oxidative decarboxylation of leucine. 4. Oxidation of L-[2-14C]leucine to 14CO2 was increased about 3-fold in acini from starved lactating rats or lactating rats fed on a 'cafeteria' diet compared with rats fed on a chow diet. Insulin decreased the formation of 14CO2 in all three situations. 5. Incorporation of L-[U-14C]- and [2-14C]-leucine into lipid was decreased in acini from starved lactating rats and lactating rats fed on a 'cafeteria' diet. Insulin tended to increase the conversion of [2-14C]leucine into lipid, but this was significant only in the case of the acini from 'cafeteria'-fed rats. 6. Experiments with (-)-**hydroxycitrate** indicate that the major route for conversion of leucine carbon into lipid in acini is via citrate translocation from the mitochondria. 7. The physiological implications of these findings are discussed.

L78 ANSWER 10 OF 47 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 82032020 MEDLINE
DOCUMENT NUMBER: 82032020 PubMed ID: 7026709
TITLE: Role of fatty acid synthesis in the control of insulin-stimulated glucose utilization by rat adipocytes.
AUTHOR: Fried S K; Lavau M; Pi-Sunyer F X
CONTRACT NUMBER: AM 26687 (NIADDK)
SOURCE: JOURNAL OF LIPID RESEARCH, (1981 Jul) 22 (5) 753-62.
Journal code: IX3; 0376606. ISSN: 0022-2275.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198112
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19811222

L78 ANSWER 11 OF 47 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 81143625 MEDLINE
DOCUMENT NUMBER: 81143625 PubMed ID: 6162901
TITLE: Sebaceous gland differentiation: III. The uses and limitations of freshly isolated mouse preputial gland cells for the in vitro study of hormone and drug action.
AUTHOR: Wheatley V R; Brind J L
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1981 Apr) 76 (4) 293-6.
Journal code: IHZ; 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198105
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810521

AB The effects of selected hormones and drugs on freshly isolated mouse preputial gland cells have been studied. The steroid hormones, testosterone, DHT, androstanediol, androsterone and androstanedione all failed to stimulate, and estradiol failed to inhibit, either DNA or lipid synthesis under the conditions studied. Thyroxine and insulin had no effect on lipogenesis but epinephrine and PGE2 caused significant stimulation as did Bt2cAMP. The antilipemic drugs clofibrate, nicotinic acid and **hydroxycitrate** were all able to inhibit lipogenesis. Of the anti-acne drugs only L-DOPA was able to inhibit lipogenesis, neither tetracycline nor trans-retinoic acid showed any effect. Pyridoxine was unable to inhibit lipogenesis but DMSO caused dramatic stimulation though it was without effect on DNA synthesis. Evidence is presented which suggests that the lack of response to steroid hormones is not due to the inability of the cells to take up and metabolize the steroids but is due to the fact that the time-span of exposure is not long enough to elicit a cellular response. It is concluded that these freshly isolated cells are suitable for the study of those effects of hormones and drugs which occur within the first 3 hr after exposure to the compound.

L78 ANSWER 12 OF 47 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 78043918 MEDLINE
DOCUMENT NUMBER: 78043918 PubMed ID: 923910
TITLE: Effects of fluoroacetate and (-)-**hydroxycitrate** on fatty acid synthesis in rat epididymal adipose tissue [proceedings].
AUTHOR: Brownsey R W; Bridges B J; Denton R M
SOURCE: BIOCHEMICAL SOCIETY TRANSACTIONS, (1977) 5 (5) 1286-8. Journal code: E48; 7506897. ISSN: 0300-5127.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197801
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19780127

L78 ANSWER 13 OF 47 MEDLINE
ACCESSION NUMBER: 2001105565 MEDLINE
DOCUMENT NUMBER: 20583412 PubMed ID: 11187927
TITLE: Dietary fat intake, supplements, and weight loss.
AUTHOR: Dyck D J
CORPORATE SOURCE: Department of Human Biology and Nutritional Sciences, University of Guelph, ON.
SOURCE: CANADIAN JOURNAL OF APPLIED PHYSIOLOGY, (2000 Dec) 25 (6) 495-523. Ref: 159
Journal code: BOT. ISSN: 1066-7814.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208

AB Although there remains controversy regarding the role of macronutrient balance in the etiology of obesity, the consumption of high-fat diets appears to be strongly implicated in its development. Evidence that fat oxidation does not adjust rapidly to acute increases in dietary fat, as well as a decreased capacity to oxidize fat in the postprandial state in the obese, suggest that diets high in fat may lead to the accumulation of

fat stores. Novel data is also presented suggesting that in rodents, high-fat diets may lead to the development of leptin resistance in skeletal muscle and subsequent accumulations of muscle triacylglycerol. Nevertheless, several current fad diets recommend drastically reduced carbohydrate intake, with a concurrent increase in fat content. Such recommendations are based on the underlying assumption that by reducing circulating insulin levels, lipolysis and lipid oxidation will be enhanced and fat storage reduced. Numerous supplements are purported to increase fat oxidation (carnitine, conjugated linoleic acid), increase metabolic rate (ephedrine, pyruvate), or inhibit hepatic lipogenesis (**hydroxycitrate**). All of these compounds are currently marketed in supplemental form to increase weight loss, but few have actually been shown to be effective in scientific studies. To date, there is little or no evidence supporting that carnitine or **hydroxycitrate** supplementation are of any value for weight loss in humans. Supplements such as pyruvate have been shown to be effective at high dosages, but there is little mechanistic information to explain its purported effect or data to indicate its effectiveness at lower dosages. Conjugated linoleic acid has been shown to stimulate fat utilization and decrease body fat content in mice but has not been tested in humans. The effects of ephedrine, in conjunction with methylxanthines and aspirin, in humans appears unequivocal but includes various cardiovascular side effects. None of these compounds have been tested for their effectiveness or safety over prolonged periods of time.

L78 ANSWER 14 OF 47 MEDLINE
ACCESSION NUMBER: 97287758 MEDLINE
DOCUMENT NUMBER: 97287758 PubMed ID: 9142886
TITLE: Malonyl-CoA regulation in skeletal muscle: its link to cell citrate and the glucose-fatty acid cycle.
AUTHOR: Saha A K; Vavvas D; Kurowski T G; Apazidis A; Witters L A; Shafrir E; Ruderman N B
CORPORATE SOURCE: Evans Department of Medicine, Boston University Medical Center, Massachusetts 02118, USA.
CONTRACT NUMBER: DK-19514 (NIDDK)
DK-49417 (NIDDK)
T-32-DK-07201 (NIDDK)
+
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1997 Apr) 272 (4 Pt 1) E641-8.
Journal code: 3U8; 0370511. ISSN: 0002-9513.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970620
Last Updated on STN: 19970620
Entered Medline: 19970606

AB Malonyl-CoA is an inhibitor of carnitine palmitoyltransferase I, the enzyme that controls the oxidation of fatty acids by regulating their transfer into the mitochondria. Despite this, knowledge of how malonyl-CoA levels are regulated in skeletal muscle, the major site of fatty acid oxidation, is limited. Two- to fivefold increases in malonyl-CoA occur in rat soleus muscles incubated with glucose or glucose plus insulin for 20 min [Saha, A. K., T. G. Kurowski, and N. B. Ruderman. Am. J. Physiol. 269 (Endocrinol. Metab. 32): E283-E289, 1995]. In addition, as reported here, acetoacetate in the presence of glucose increases malonyl-CoA levels in the incubated soleus. The increases in malonyl-CoA in all of these situations correlated closely with increases in the concentration of citrate ($r2 = 0.64$) and to an even greater extent the sum of citrate plus malate ($r2 = 0.90$), an antiporter for citrate efflux from the mitochondria. Where measured, no increase in the activity of acetyl-CoA

carboxylase (ACC) was found. Inhibition of ATP citrate lyase with **hydroxycitrate** markedly diminished the increases in malonyl-CoA in these muscles, indicating that citrate was the major substrate for the malonyl-CoA precursor, cytosolic acetyl-CoA. Studies with enzyme purified by immunoprecipitation indicated that the observed increases in citrate could have also allosterically activated ACC. The results suggest that in the presence of glucose, insulin and acetoacetate acutely increase malonyl-CoA levels in the incubated soleus by increasing the cytosolic concentration of citrate. This novel mechanism could complement the glucose-fatty acid cycle in determining how muscle chooses its fuels. It could also provide a means by which glucose acutely modulates signal transduction in muscle and other cells (e.g., the pancreatic beta-cell) in which its metabolism is determined by substrate availability.

L78 ANSWER 15 OF 47 MEDLINE
ACCESSION NUMBER: 94283727 MEDLINE
DOCUMENT NUMBER: 94283727 PubMed ID: 8013751
TITLE: More direct evidence for a malonyl-CoA-carnitine palmitoyltransferase I interaction as a key event in pancreatic beta-cell signaling.
AUTHOR: Chen S; Ogawa A; Ohneda M; Unger R H; Foster D W; McGarry J D
CORPORATE SOURCE: Department of Internal Medicine, Gifford Laboratories, University of Texas Southwestern Medical Center at Dallas 75235-8858.
CONTRACT NUMBER: DK-18575 (NIDDK)
DK-42582 (NIDDK)
SOURCE: DIABETES, (1994 Jul) 43 (7) 878-83.
Journal code: E8X; 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940810
Last Updated on STN: 19980206
Entered Medline: 19940725

AB We sought to explore the emerging concept that malonyl-CoA generation, with concomitant suppression of mitochondrial carnitine palmitoyltransferase I (CPT I), represents an important component of glucose-stimulated insulin secretion (GSIS) by the pancreatic beta-cell (Prentki M, Vischer S, Glennon MC, Regazzi R, Deeney JT, Corkey BE: Malonyl-CoA and long-chain acyl-CoA esters as metabolic coupling factors in nutrient-induced insulin secretion. J Biol Chem 267:5802-5810, 1992). Accordingly, pancreases from fed rats were perfused with basal (3 mM) followed by high (20 mM) glucose in the absence or presence of 2 mM **hydroxycitrate** (HC), an inhibitor of ATP-citrate (CIT) lyase (the penultimate step in the glucose-->malonyl-CoA conversion). HC profoundly inhibited GSIS, whereas CIT had no effect. Inclusion of 0.5 mM palmitate in the perfusate significantly enhanced GSIS and completely offset the negative effect of HC. In isolated islets, HC stimulated [1-14C]palmitate oxidation in the presence of basal glucose and markedly obtunded the inhibitory effect of high glucose. Directional changes in 14C incorporation into phospholipids were opposite to those of 14CO2 production. At a concentration of 0.2 mM, 2-bromostearate, 2-bromopalmitate and etomoxir (all CPT I inhibitors) potentiated GSIS by the pancreas and inhibited palmitate oxidation in islets. However, at 0.05 mM, etomoxir did not influence insulin secretion but still caused significant suppression of fatty acid oxidation. The results provide more direct evidence for a pivotal role of malonyl-CoA suppression of CPT I, with attendant elevation of the cytosolic long-chain acyl-CoA concentration, in GSIS from the normal pancreatic beta-cell.(ABSTRACT TRUNCATED AT 250 WORDS)

L78 ANSWER 16 OF 47 MEDLINE
ACCESSION NUMBER: 83073434 MEDLINE
DOCUMENT NUMBER: 83073434 PubMed ID: 6756313
TITLE: Mechanism of the control of pulmonary and hepatic fatty acid synthesis by the thyroid hormones.
AUTHOR: Das D K; Ganguly M
SOURCE: ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1982 Oct 1) 218 (1) 142-55.
Journal code: 6SK; 0372430. ISSN: 0003-9861.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198301
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19830107

L78 ANSWER 17 OF 47 IPA COPYRIGHT 2002 ASHP

ACCESSION NUMBER: 2000:3595 IPA
DOCUMENT NUMBER: 37-03595
TITLE: Safe dieting. Part 1. Creating a comprehensive weight management center
AUTHOR: Eaton, J.
CORPORATE SOURCE: New York Nutrition Network of New York City, New York, NY, USA
SOURCE: Natural Pharmacy (USA), (Jan 1999) Vol. 3, pp. 1, 12-13. 6 Refs.
ISSN: 1089-4853.
DOCUMENT TYPE: Journal
FILE SEGMENT: HUMAN
LANGUAGE: English
AB Safe natural alternatives to prescription medications for weight loss management are described, including L-carnitine, **hydroxycitric acid**, poliglusam (chitosan), collagen, and 7-keto-DHEA.
M. Therese Gyi

L78 ANSWER 18 OF 47 IPA COPYRIGHT 2002 ASHP

ACCESSION NUMBER: 1999:3246 IPA
DOCUMENT NUMBER: 36-04478
TITLE: Garcinia cambogia (**hydroxycitric acid**) as a potential antiobesity agent: randomized controlled trial
AUTHOR: Heymsfield, S. B.; Allison, D. B.; Vasselli, J. R.; Pietrobelli, A.; Nunez, C.; et al
CORPORATE SOURCE: Obesity Res. Ctr., 1090 Amsterdam Ave., 14th Fl., New York, NY 10025, USA Internet: sbh2@columbia.edu
SOURCE: Journal of the American Medical Association (USA), (Nov 11 1998) Vol. 280, pp. 1596-1600. 32 Refs.
CODEN: JAMAAP; ISSN: 0098-7484.
DOCUMENT TYPE: Journal
FILE SEGMENT: HUMAN
LANGUAGE: English
AB A double blind, placebo controlled study evaluating **hydroxycitric acid**, the active component of Garcinia cambogia, in weight loss and fat mass loss was conducted in 135 overweight patients, ages 18-65 yr, of whom 69 received a placebo and 66 received 500 mg of **hydroxycitric acid** contained in two 500 mg caplets of G. cambogia extract 3 times daily before meals; all patients also received dietary instruction and were followed up for 12 wk.

Forty-two patients in the active treatment group and 42 in the placebo group completed the study. Patients in both groups lost a significant amount of weight; however, between-group weight loss differences were not significant. There were no significant differences in estimated body fat mass loss between treatment groups, and the fraction of subject weight loss as fat was not influenced by treatment.
Peggy L. Ruppel

L78 ANSWER 19 OF 47 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
ACCESSION NUMBER: 2001:224396 CAPLUS
DOCUMENT NUMBER: 134:256874
TITLE: Methods and pharmaceutical preparations for improving glucose metabolism with (-)-**hydroxycitric acid**
INVENTOR(S): Clouatre, Dallas L.; Dunn, James M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 4 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6207714	B1	20010327	US 2000-661588	20000914

PRIORITY APPLN. INFO.: US 1999-153840 P 19990914

AB Disclosed is a method whereby the glucose metab. in an individual showing evidence of dysregulation, as is found in **insulin** resistance, reactive hyperglycemia and/or elevated blood sugar levels and/or diabetes, is improved when that person receives an appropriate oral administration of (-)-**hydroxycitric acid**. The potassium salt of (-)-**hydroxycitric acid** is the preferred form of the compd., followed by the sodium salt. The regulation of glucose levels over any given period of time may be improved with a controlled release form of (-)-**hydroxycitric acid**. Controlled release can be used to provide a sustained and modulated amt. of the active to the body as desired and therefore regulate the use of the compd. as a hypoglycemic agent.

IT **185196-38-7**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(improving glucose metab. with (-)-**hydroxycitric acid** and its salts)

IT **27750-10-3**, (-)-**Hydroxycitric acid** **64913-19-5 132436-67-0 213385-58-1**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(improving glucose metab. with (-)-**hydroxycitric acid** and its salts)

REFERENCE COUNT: 4
REFERENCE(S):
(1) Hastings; US 5626849 1997 CAPLUS
(2) Lowenstein; US 3764692 1973 CAPLUS
(3) Majeed; US 5783603 1998 CAPLUS
(4) McCarty; US 5914326 1999

L78 ANSWER 20 OF 47 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:851796 CAPLUS
DOCUMENT NUMBER: 135:366751
TITLE: Methods and pharmaceutical preparations for normalizing blood pressure with (-)-**hydroxycitric acid**
INVENTOR(S): Clouatre, Dallas L.; Dunn, James M.
PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 6 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2001044469	A1	20011122	US 2001-781491	20010213

PRIORITY APPLN. INFO.: US 2000-181285 P 20000209

AB A method whereby the blood pressure metab. in an individual showing evidence of dysregulation is improved when that person receives an appropriate oral administration of (-)-**hydroxycitric acid** (I) . The potassium salt of I is a preferred form of the compd., followed by the sodium salt, then by the amide and other derivs. of the acid. The regulation of blood pressure levels over any given period of time may be improved with a controlled release form of I. Controlled release can be used to provide a sustained and modulated amt. of the active to the body as desired and therefore regulate the use of the compd. as a hypotensive agent. Oral administration of 3-4 g of potassium salt of I per day in two divided doses in extremely obese patients normalized the blood pressure along with decrease of blood glucose level.

IT **27750-10-3**, (-)-**Hydroxycitric acid**
27750-10-3D, (-)-**Hydroxycitric acid**, alk. earth metal salts **64913-19-5 132436-67-0 185196-38-7 213385-58-1**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods and pharmaceutical prepns. for normalizing blood pressure with **hydroxycitric acid** salts)

L78 ANSWER 21 OF 47 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:513666 CAPLUS
DOCUMENT NUMBER: 135:256592
TITLE: The effects of 2-week ingestion of (-)-hydroxycitrate and (-)-hydroxycitrate combined with medium-chain triglycerides on satiety, fat oxidation, energy expenditure and body weight
AUTHOR(S): Kovacs, E. M. R.; Westerterp-Plantenga, M. S.; Saris, W. H. M.
CORPORATE SOURCE: Department of Human Biology, Maastricht University, Maastricht, 6200 MD, Neth.
SOURCE: Int. J. Obes. (2001), 25(7), 1087-1094
CODEN: IJOBDP; ISSN: 0307-0565
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of 2-wk dietary supplementation with (-)-hydroxycitrate (HCA) and HCA plus medium-chain triglycerides (MCT) on satiety, fat oxidn., energy expenditure (EE), and body wt. (BW) loss were examd. in 11 obese men (age 47.+-.16 yr; body mass index 27.4.+-.8.2 kg/m2) in three 2-wk intervention periods sepd. by 4-wk washout periods. The men consumed 3 self-selected meals and 4 iso-energetic (420 kJ) snacks daily with no supplement (PLA), 500 mg HCA, or 500 mg HCA plys 3 g MCT. Each intervention period ended with a 36-h stay in the respiration chamber. There was BW loss during the 2-wk intervention (PLA -1.0.+-.0.4 kg; HCA -1.5.+-.0.5 kg; HCA + MCT -1.3.+-.0.2 kg), but the decreases were not different among the 3 treatments. The 24-h EE (PLA 11.8.+-.0.2 MJ; HCA 11.7.+-.0.1 MJ; HCA + MCT 11.5.+-.0.1 MJ), 24-h RQ (0.85.+-.0.00 in all 3 treatments), and the area under the curve of appetite-related parameters were not different among the 3 treatments. Thus, 2-wk supplementation with HCA and HCA plus MCT did not increase satiety, fat oxidn., 24-h EE,

or BW loss compared to PLA in men losing BW.
IT **9004-10-8, Insulin,** biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(dietary (-)-hydroxycitrate alone or with medium-chain triglycerides effects on satiety, fat oxidn., energy expenditure and body wt. in obese men)
IT **27750-10-3, (-)-Hydroxycitric acid**
RL: FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary (-)-hydroxycitrate alone or with medium-chain triglycerides effects on satiety, fat oxidn., energy expenditure and body wt. in obese men)
REFERENCE COUNT: 60
REFERENCE(S): (1) Bach, A; Am J Clin Nutr 1982, V36, P950 CAPLUS
(2) Bach, A; J Lipid Res 1996, V37, P708 CAPLUS
(6) Bremer, J; Physiol Rev 1983, V63, P1420 CAPLUS
(11) Flatt, J; J Clin Invest 1985, V76, P1019 CAPLUS
(13) Furuse, M; Physiol Behav 1992, V52, P815 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L78 ANSWER 22 OF 47 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:336612 CAPLUS
DOCUMENT NUMBER: 133:119495
TITLE: Toward a wholly nutritional therapy for type 2 diabetes
AUTHOR(S): McCarty, M. F.
CORPORATE SOURCE: Helicon Foundation, San Diego, CA, USA
SOURCE: Med. Hypotheses (2000), 54(3), 483-487
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 84 refs. is given. It may now be feasible to target specific supplemental nutrients to each of the key dysfunctions which conspire to maintain hyperglycemia in type 2 diabetes: bioactive chromium for skeletal muscle **insulin** resistance, conjugated linoleic acid for adipocyte **insulin** resistance, high-dose biotin for excessive hepatic glucose output, and coenzyme Q10 for beta cell failure. Nutritional strategies which disinhibit hepatic fatty acid oxidn. (involving hydroxycitrate, carnitine, pyruvate, and other adjuvants) may likewise prove beneficial - in the short term, by decreasing serum free fatty acids and, in the longer term, by promoting regression of visceral obesity. The nutrients and food factors recommended here appear to be safe and well tolerated, and thus may have particular utility for diabetes prevention.
IT **27750-10-3, Hydroxycitric acid**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(toward a wholly nutritional therapy for type 2 diabetes)
REFERENCE COUNT: 84
REFERENCE(S): (1) Anderson, R; Diabetes 1997, V46, P1786 CAPLUS
(2) Anderson, R; J Agric Food Chem 1978, V26, P1219 CAPLUS
(7) Belury, M; J Nutr Biochem 1997, V8, P579 CAPLUS
(8) Berry, M; Eur J Biochem 1983, V131, P205 CAPLUS
(9) Berry, M; Metabolism 1985, V34, P141 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L78 ANSWER 23 OF 47 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:268507 CAPLUS
DOCUMENT NUMBER: 128:278299
TITLE: Magnesium (-)-hydroxycitrate, method of preparation, applications, and compositions, in particular pharmaceutical, containing same

INVENTOR(S): Shrivastava, Ravi; Lambropoulos, Patrick
PATENT ASSIGNEE(S): Shrivastava, Ravi, Fr.; Lambropoulos, Patrick
SOURCE: PCT Int. Appl., 23 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9817671	A1	19980430	WO 1997-FR1860	19971017
W: AU, CA, JP, KR, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
FR 2754820	A1	19980424	FR 1996-13094	19961022
FR 2754820	B1	19991022		
AU 9748717	A1	19980515	AU 1997-48717	19971017
AU 717533	B2	20000330		
EP 937085	A1	19990825	EP 1997-911285	19971017
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001503744	T2	20010321	JP 1998-519029	19971017
KR 2000052687	A	20000825	KR 1999-703474	19990421
US 6221901	B1	20010424	US 1999-284864	19990422

PRIORITY APPLN. INFO.: FR 1996-13094 A 19961022
WO 1997-FR1860 W 19971017

AB The invention concerns magnesium (-)-hydroxycitrate, its method of prepn., its applications in dietetics and in therapeutics particularly in the cardiovascular field, and pharmaceutical compns. contg. it. Thus, magnesium (-)-hydroxycitrate is prepd. from reaction of an ext. of Garcinia cambogia with an aliph. alc. (e.g., EtOH) to obtain a ppt. which is treated with a tannin fixative (e.g., poly(vinylpyrrolidone)), filtered, and the remaining soln. agitated with an anion exchange resin, the supernatant is eliminated, and the product is eluted and dried. Magnesium (-)-hydroxycitrate is useful in the therapeutic treatment of cardiovascular diseases. The antioxidant and antihypertensive activities of the (-)-hydroxycitrate in rat, its antihypercholesterolemic and antiatherosclerotic activities in rabbit, and the toxicity in rat are reported. An assocn. of magnesium (-)-hydroxycitrate with Mg, Cu, Co, Zn, Ni, Se, Si, Mn, Li, or Fe, ionized or not, and at least one vitamin is claimed. Pharmaceutical formulations contg. magnesium (-)-hydroxycitrate are claimed (6 examples). Magnesium (-)-hydroxycitrate or an assocd. compd. described above are applicable to dietetic/nutritional or cosmetic products.

IT **132436-67-0P**
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of magnesium (-)-hydroxycitrate for treatment of cardiovascular diseases)

L78 ANSWER 24 OF 47 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:118607 CAPLUS
DOCUMENT NUMBER: 128:149592
TITLE: Method of treatment for carbohydrate addiction with anorexients
INVENTOR(S): Bernstein, Richard K.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 8 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5716976	A	19980210	US 1996-615616	19960313

AB A method is described for alleviating carbohydrate addiction by administration of anorexients on a schedule that avoids tolerance to the anorexient.

IT **27750-10-3, Hydroxycitric acid**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(anorexient treatment of carbohydrate addiction)

L78 ANSWER 25 OF 47 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:784225 CAPLUS
DOCUMENT NUMBER: 130:177001
TITLE: Utility of metformin as an adjunct to hydroxycitrate/carnitine for reducing body fat in diabetics
AUTHOR(S): McCarty, M. F.
CORPORATE SOURCE: Nutrition 21, San Diego, CA, 92109, USA
SOURCE: Med. Hypotheses (1998), 51(5), 399-403
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 39 refs. Excessive exposure of tissues to fatty acids is likely to be the chief cause of the various dysfunctions that lead to sustained hyperglycemia in type II diabetes. These dysfunctions are likely to be substantially reversible if body fat and dietary fat can be greatly reduced. Disinhibition of hepatic fatty acid oxidn. with hydroxycitrate (HCA) and carnitine has considerable potential as a new wt.-loss strategy, but in diabetics runs the risk of further enhancing excessive hepatic gluconeogenesis. Since the clin. utility of metformin in diabetes is probably traceable to inhibition of gluconeogenesis, its use as an adjunct to HCA/carnitine treatment of obesity in diabetics deserves evaluation, particularly as metformin therapy itself tends to reduce body wt. A consideration of relevant evidence suggests that metformin therapy will not impede the activation of fatty acid oxidn. by HCA/carnitine, and is likely to potentiate the appetite-suppressant and thermogenic benefits of this strategy. Indeed, since metformin has been reported to lower body wt. and improve cardiovascular risk factors in obese non-diabetics, a broader application of a metformin/HCA/carnitine therapy for obesity can be contemplated.

IT **27750-10-3, Hydroxycitric acid**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(utility of metformin as an adjunct to hydroxycitrate/carnitine for reducing body fat in diabetics)

REFERENCE COUNT: 78
REFERENCE(S):
(1) Argaud, D; Eur J Biochem 1993, V213, P1341 CAPLUS
(7) Bolinder, J; Diabetes 1983, V32, P117 CAPLUS
(8) Chen, Y; J Clin Endocrinol Metab 1987, V64, P17 CAPLUS
(11) Cortez, M; Am J Clin Nutr 1991, V53, P847 CAPLUS
(12) DeRubertis, F; Diabetes 1994, V43, P1 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L78 ANSWER 26 OF 47 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1985:209206 BIOSIS
DOCUMENT NUMBER: BR29:99202
TITLE: VARIATIONS IN HEPATIC STEROL SYNTHESIS AND TRITIATED WATER INCORPORATION EFFECTS OF LIPOGENIC PRECURSORS PANCREATIC

HORMONES AND DRUGS.
AUTHOR(S): BJORNSSON O G; PULLINGER C R; GIBBONS G F
CORPORATE SOURCE: MRC LIPID METAB. UNIT, HAMMERSMITH HOSP., UK.
SOURCE: 19TH ANNUAL MEETING OF THE EUROPEAN SOCIETY FOR CLINICAL INVESTIGATION, TOULOUSE, FRANCE, APR. 24-27, 1985. EUR J CLIN INVEST, (1985) 15 (2 PART 2), A3.
CODEN: EJCIB8. ISSN: 0014-2972.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L78 ANSWER 27 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001312864 EMBASE
TITLE: Hepatothermic therapy of obesity: Rationale and an inventory of resources.
AUTHOR: McCarty M.F.
CORPORATE SOURCE: M.F. McCarty, Pantox Laboratories, 4622 Santa Fe Street, San Diego, CA 92109, United States
SOURCE: Medical Hypotheses, (2001) 57/3 (324-336).
Refs: 169
ISSN: 0306-9877 CODEN: MEHYDY
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
017 Public Health, Social Medicine and Epidemiology
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Hepatothermic therapy (HT) of obesity is rooted in the observation that the liver has substantial capacities for both fatty acid oxidation and for thermogenesis. When hepatic fatty acid oxidation is optimized, the newly available free energy may be able to drive hepatic thermogenesis, such that respiratory quotient declines while basal metabolic rate increases, a circumstance evidently favorable for fat loss. Effective implementation of HT may require activation of carnitine palmitoyl transferase-1 (rate-limiting for fatty acid beta-oxidation), an increase in mitochondrial oxaloacetate production (required for optimal Krebs cycle activity), and up-regulation of hepatic thermogenic pathways. The possible utility of various natural agents and drugs for achieving these objectives is discussed. Potential components of HT regimens include EPA-rich fish oil, sesamin, hydroxycitrate, pantethine, L-carnitine, pyruvate, aspartate, chromium, coenzyme Q10, green tea polyphenols, conjugated linoleic acids, DHEA derivatives, cilostazol, diazoxide, and fibrate drugs. Aerobic exercise training and very-low-fat, low-glycemic-index, high-protein or vegan food choices may help to establish the hormonal environment conducive to effective HT. High-dose biotin and/or metformin may help to prevent an excessive increase in hepatic glucose output. Since many of the agents contemplated as components of HT regimens are nutritional or food-derived compounds likely to be health protective, HT is envisioned as an on-going lifestyle rather than as a temporary 'quick fix'. Initial clinical efforts to evaluate the potential of HT are now in progress. .COPYRGT. 2001 Harcourt Publishers Ltd.

L78 ANSWER 28 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001379532 EMBASE
TITLE: Anabolic steroid misuse: How much should we know?.
AUTHOR: Gonzalez A.; McLachlan S.; Keaney F.
CORPORATE SOURCE: Dr. A. Gonzalez, Psychiatry Rehabilitation Department, Harplands Hospital, North Staffordshire Combined NHS, Harpfields Road, Stoke-on-Trent ST4 GRR, United Kingdom
SOURCE: International Journal of Psychiatry in Clinical Practice, (2001) 5/3 (159-167).

Refs: 63
ISSN: 1365-1501 CODEN: IJPCFZ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
040 Drug Dependence, Alcohol Abuse and Alcoholism
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The misuse of anabolic androgenic steroids (AAS) and other performance enhancing (ergogenic) drugs remains largely unrecognized by many health professionals. The real extent of the problem is unknown, probably as a result of a combination of various methodological difficulties. Examples include poor definition of cases, obstacle in recruiting large enough samples for longitudinal follow-up, ethical issues as AAS are obtained from the black market and the covert nature of the problem itself. Our review attempts to alert psychiatrists and mental health professionals to the risks associated with these compounds. We cover the pharmacology, epidemiology, the use and misuse and relevant complications.

L78 ANSWER 29 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000400228 EMBASE
TITLE: A randomized, double-blind, placebo-controlled trial of a new weight-reducing agent of natural origin.
AUTHOR: Thom E.
CORPORATE SOURCE: Dr. E. Thom, Parexel Medstat AS, PO Box 210, N-2001 Lillestrom, Norway. erling.thom@parexel.com
SOURCE: Journal of International Medical Research, (2000) 28/5 (229-233).
Refs: 13
ISSN: 0300-0605 CODEN: JIMRBV
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 006 Internal Medicine
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The efficacy and tolerability of a new weight-reduction agent, based on natural ingredients, was investigated in this randomized, placebo-controlled, double-blind study. The product reduces the absorption of different types of sugar from the gastrointestinal tract. Forty obese volunteers were included in the 12-week study. Body weight, body composition and blood pressure were recorded at baseline and every month during the study. The results show a significant difference in weight reduction in favour of the active group (3.5 kg versus 1.2 kg). Body composition measurements showed that > 85% of the reduction in the active group is fat loss. The tolerability was similar and good in both groups. This product shows promising results and should be studied more extensively at different dose levels.

L78 ANSWER 30 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999281025 EMBASE
TITLE: (-)-Hydroxycitric acid does not affect energy expenditure and substrate oxidation in adult males in a post-absorptive state.
AUTHOR: Kriketos A.D.; Thompson H.R.; Greene H.; Hill J.O.
CORPORATE SOURCE: Dr. J.O. Hill, Center for Human Nutrition, Univ. CO Health Sciences Center, Campus Box C225, 4200 East Ninth Avenue, Denver, CO 80262, United States

SOURCE: International Journal of Obesity, (1999) 23/8 (867-873).
Refs: 27
ISSN: 0307-0565 CODEN: IJOBDP
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB OBJECTIVE: (-)-Hydroxycitric acid ((-)-HCA) is available as a herbal supplement, and promoted as a weight loss agent. It is hypothesized that (-)-HCA can increase fat oxidation by inhibiting citrate lyase, an enzyme which plays a crucial role in energy metabolism during de novo lipogenesis. The indirect inhibition of the cytosolic pool of citrate by [-)-HCA and the subsequent reduction in acetyl coenzyme A and oxaloacetate alters steps in the citric acid cycle that promote fat oxidation. The objective of this study was to determine the effect of (-)-HCA on marker substrates of altered metabolism, as well as on respiratory quotient (RO) and energy expenditure (EE) in humans, following an overnight fast and during a bout of exercise. HYPOTHESIS OF STUDY: We hypothesized that supplementation with (-)-HCA would result in an increase in fat oxidation and metabolic rate, reflected by an increase in .beta.-hydroxybutyrate and EE and/or a decrease in RQ. Furthermore, during moderately intense exercise, we hypothesized that (-)-HCA supplementation would increase the rate of lactate conversion to glucose in the liver, with a subsequent reduction of circulating lactate and an elevation of circulating ketone bodies due to the increased partial oxidation of fatty acids (FA) in mitochondria. Studies have examined the fat regulating action of (-)-HCA on steps of the citric acid cycle in rodents showing reductions in body weight and food intake. No studies have investigated the effects of (-)-HCA supplementation in conjunction with a typical daily dietary composition (that is approx 30-35% fat) on metabolic processes which could influence body weight regulation in humans. DESIGN: This was a double blind, placebo controlled, randomized, crossover study involving three days of (-)-HCA (3.0 g/d) or placebo supplementation. The effects of (-)-HCA supplementation on metabolic parameters with or without moderately intense exercise was studied over four laboratory visits. SUBJECTS: Sedentary adult male subjects (n = 10, age: 22-38 y, body mass index (BMI) 22.4-37.6 kg/m2). MEASUREMENTS: Two of the four visits involved no exercise (Protocol A) with and without (-)-HCA treatment, while the remaining two visits included a moderately intense exercise bout [Protocol B; 30 min at 40% maximal aerobic fitness (V2max) and 15 min at 60% VO2max) with and without (-)-HCA treatment. EE (by indirect calorimetry) and RQ were measured for 150 min following an overnight fast. Blood samples were collected for the determination of glucose, insulin, glucagon, lactate, and .beta.-hydroxybutyrate concentrations. RESULTS: In a fasted state and following 3 d of (-)-HCA treatment, RQ was not significantly lowered during rest (Protocol A) nor during exercise (Protocol B) compared with the placebo treatment. Treatment with (-)-HCA did not affect EE, either during rest or during moderately intense exercise. Furthermore, the blood substrates measured were not significantly different between treatment groups under the fasting conditions of this study. CONCLUSION: These results do not support the hypothesis that (-)-HCA alters the short-term rate of fat oxidation in the fasting state during rest or moderate exercise, with doses likely to be achieved in humans while subjects maintain a typical Western diet (approx 30-35% total calories as fat).

L78 ANSWER 31 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000298826 EMBASE
TITLE: Current and potential drugs for treatment of obesity.
AUTHOR: Bray G.A.; Greenway F.L.
CORPORATE SOURCE: Dr. G.A. Bray, 6400 Perkins Road, Baton Rouge, LA 70808, United States

SOURCE: Endocrine Reviews, (1999) 20/6 (805-875).
Refs: 999
ISSN: 0163-769X CODEN: ERVIDP
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English

L78 ANSWER 32 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 83160847 EMBASE
DOCUMENT NUMBER: 1983160847
TITLE: The role of substrate supply in the regulation of cholesterol biosynthesis in rat hepatocytes.
AUTHOR: Pullinger C.R.; Gibbons G.F.
CORPORATE SOURCE: Med. Res. Counc. Lipid Metab. Unit, Hammersmith Hosp., London W12 0HS, United Kingdom
SOURCE: Biochemical Journal, (1983) 210/3 (625-632).
CODEN: BIJOAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
AB Compactin, (-)-hydroxycitrate and dexamethasone gave rise to a decrease in the rate of cholesterol production in hepatocytes from fed rats by interfering with the flow of substrate into the sterol biosynthetic pathway. The cells responded to the deficit of biosynthetic sterol by increasing the activity of hydroxymethylglutaryl-CoA reductase (HMG-CoA reductase). Compactin and (-)-hydroxycitrate gave similar results in hepatocytes from rats starved for 24 h but in this case dexamethasone had no significant effect. Exogenous oleate interferes with the production of carbohydrate-derived acetyl-CoA and also gives rise initially to opposing effects on the rate of sterol synthesis and HMG-CoA reductase activity. Over a longer period, however, oleate itself was capable of replacing carbohydrate as the major source of carbon for sterol synthesis. The increase in HMG-CoA reductase activity observed when liver cells were incubated in the presence of compactin, (-)-hydroxycitrate or oleate could be partially reversed by the simultaneous presence of glucagon. Under some physiological conditions, a deficiency of biosynthetic cholesterol or of a related precursor may lead to an increase in the activity of HMG-CoA reductase.

L78 ANSWER 33 OF 47 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 82239508 EMBASE
DOCUMENT NUMBER: 1982239508
TITLE: Effects of insulin on glucose utilization and lipogenesis in interscapular brown adipose tissue and liver of the rat: Possible sites of insulin action.
AUTHOR: Sugden M.C.; Marshall C.E.; Watts D.I.
CORPORATE SOURCE: Dept. Biochem., Charing Cross Hosp. Med. Sch., London, United Kingdom
SOURCE: Diabetologia, (1982) 23/2 (No 301).
CODEN: DBTGAJ
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English

L78 ANSWER 34 OF 47 DRUGU COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1991-23981 DRUGU P
TITLE: Pharmacological Approaches to Appetite Suppression.

AUTHOR: Blundell J
LOCATION: Leeds, United Kingdom
SOURCE: Trends Pharmacol.Sci. (12, No. 4, 147-57, 1991)
CODEN: TPHSDY ISSN: 0165-6147
AVAIL. OF DOC.: University of Leeds, Leeds LS2 9JT, England.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB Pharmacological approaches to appetite suppression are reviewed. The nature of the appetite control system, pharmacological targets forappetite control, peripherally and centrally active agents are discussed. Qualitative aspects of appetite, and the possibility of pharmacological control are considered.

L78 ANSWER 35 OF 47 DRUGU COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1988-17914 DRUGU P
TITLE: Experimental Manipulations of Eating: Advances in Animal Models for Study Anorectic Agents.
AUTHOR: Blundell J E; Thurlby P L
LOCATION: Milan, Italy
SOURCE: Pharmacol.Ther. (34, No. 3, 349-401, 1987) 4 Fig. 4 Tab. 480 Ref.
CODEN: PHTHDT ISSN: 0163-7258
AVAIL. OF DOC.: Biopsychology Group, Psychology Department, University of Leeds, Leeds LS2 9JT, England.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB The range of procedures and pharmacological agents which have the capacity to modify eating behavior is reviewed. A number of agents suppress intake of food including glucose, lisuride, tryptophan, cocaine, muscimol, salbutamol and atropine and drugs which enhance food intake are also common. In fact, an increase in body weight is a common side effect associated with treatment with benzodiazepines such as diazepam, chlordiazepoxide and nitrazepam. Amphetamine has ambivalent effects on eating behaviour.

L78 ANSWER 36 OF 47 DRUGU COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1985-17684 DRUGU P T N S
TITLE: Mechanisms of Appetite Modulation by Drugs.
AUTHOR: Sullivan A C; Gruen R K
CORPORATE SOURCE: Roche
LOCATION: Nutley, New Jersey, United States
SOURCE: Fed.Proc.Fed.Am.Soc.Exp.Biol. (44, No. 1, Pt. 1, 139-44, 1985) 1 Fig. 2 Tab. 51 Ref.
CODEN: FEPRA7
AVAIL. OF DOC.: Department of Pharmacology II, Hoffmann-La Roche, Inc., Nutley, New Jersey 07110, U.S.A.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB The regulation of appetite is a complex process involving both central and peripheral components, and requires integration by the brain of a variety of signals from peripheral organs transmitted by neurotransmitters such as serotonin (5HT), norepinephrine (NE), GABA and dopamine (DA), peptides, hormones, including cholecystokinin (CCK), and metabolites. All currently available anorectic drugs act by central mechanisms, and have several disadvantages including limited effectiveness, side effects on the CNS, the development of tolerance, abuse potential, and rebound hyperphagia on withdrawal. Several newer

agents studied in animals appear to act peripherally, without inducing tolerance or rebound hyperphagia, and these substances may provide better appetite control for obese subjects in the future.

L78 ANSWER 37 OF 47 DRUGU COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1985-23434 DRUGU P B E
TITLE: Variations in Hepatic Sterol Synthesis and (3H)Water Incorporation: Effects of Lipogenic Precursors, Pancreatic Hormones and Drugs.
AUTHOR: Bjoernsson O G; Pullinger C R; Gibbons G F
LOCATION: London, United Kingdom
SOURCE: Eur.J.Clin.Invest. (15, No. 2, Pt. 2, A3, 1985) 2 Ref.
CODEN: EJCIB8 ISSN: 0014-2972
AVAIL. OF DOC.: MRC Lipid Metabolism Unit, Hammersmith Hospital, London, England.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT; MPC
FILE SEGMENT: Literature
AB The effects of pyruvate, lactate, glucagon, **hydroxycitrate**, **insulin**, compactin, dexamethasone and cyanohydroxycinnamate on tritium and carbon incorporation into cholesterol were studied in rat hepatocytes in vitro. (congress abstract).

L78 ANSWER 38 OF 47 DRUGU COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1983-41979 DRUGU P E
TITLE: Neuroregulators and Feeding: Implications for the Pharmacological Manipulation of Hunger and Appetite.
AUTHOR: Blundell J E
LOCATION: Leeds, United Kingdom; Milan, Italy
SOURCE: Rev.Pure Appl.Pharmacol.Sci. (3, No. 4, 381-462, 1982) 9 Fig. 1 Tab. 318 Ref.
CODEN: RPASDB
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AB The role of neurotransmitters in feeding, and the implications for the pharmacological manipulation of hunger and appetite, are reviewed.

L78 ANSWER 39 OF 47 WPIDS COPYRIGHT 2002 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1998-252672 [23] WPIDS
DOC. NO. CPI: C1998-078754
TITLE: Dietary composition comprises chitosan and vitamin C - used to decrease body weight and control hyper-cholesterolemia and hyperglycaemia.
DERWENT CLASS: B03 B04 B05 D13
INVENTOR(S): LITTERA, R
PATENT ASSIGNEE(S): (SIRC-N) SIRC NATURAL & DIETETIC FOODS SPA
COUNTRY COUNT: 23
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 841011	A1	19980513	(199823)*	EN	9
R: AL AT BE CH DE DK ES FI FR GB GR IE IT LI LT LU LV MC NL PT RO SE SI					
IT 1285809	B	19980624	(200030)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

EP 841011	A1	EP 1997-830530	19971022
IT 1285809	B	IT 1996-RM720	19961023

PRIORITY APPLN. INFO: IT 1996-RM720 19961023
AB EP 841011 A UPAB: 19980610
Dietary composition (I), comprising vitamin C and chitosan and optionally garcinia **hydroxycitrate**, organic chromium and vanadium.
USE - (I) is used to treat the overweight and obese (claimed), by lowering lipid absorption. It also stabilises sugar metabolism and treats **hyperinsulinaemia**.
ADVANTAGE - Vitamin C increases the effectiveness of chitosan as a fat binding agent. The organic chromium, vanadium and garcinia **hydroxycitrate** synergistically stabilise glucide and lipid metabolism.
Dwg.0/4

L78 ANSWER 40 OF 47 ADISALERTS COPYRIGHT 2002 (ADIS)
ACCESSION NUMBER: 2000:23777 ADISALERTS
DOCUMENT NUMBER: 800849923
TITLE: A randomized, double-blind, placebo-controlled trial of a new weight-reducing agent of natural origin
ADIS TITLE: Herbal medicines: therapeutic use.; Obesity
AUTHOR: Thom E
CORPORATE SOURCE: Parexel Medstat, Lillestrom, Norway
SOURCE: Journal of International Medical Research J Int Med Res 28: 229 233, Sep Oct 2000. (Sep 1, 2000)
DOCUMENT TYPE: (Clinical study)
REFERENCE: Obesity (Summary): Alert no. 12, 2000
FILE SEGMENT: Summary
LANGUAGE: English
WORD COUNT: 393

L78 ANSWER 41 OF 47 USPATFULL
ACCESSION NUMBER: 2001:212472 USPATFULL
TITLE: Methods and pharmaceutical preparations for normalizing **blood pressure** with (-)-**hydroxycitric acid**
INVENTOR(S): Clouatre, Dallas L., Menlo Park, CA, United States
Dunn, James M., Littleton, CO, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001044469	A1	20011122
APPLICATION INFO.:	US 2001-781491	A1	20010213 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-181285	20000209 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Dallas L. Clouatre, #357, 555 BRYANT ST., Palo Alto, CA, 94301
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 527
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method whereby the **blood pressure** metabolism in an individual showing evidence of dysregulation is improved when that person receives an appropriate oral administration of (-)-**hydroxycitric acid**. The potassium salt of (-)-**hydroxycitric acid** is a preferred form of the compound, followed by the sodium salt, then by the amide and other

derivatives of the acid. The regulation of **blood pressure** levels over any given period of time may be improved with a controlled release form of (-)-**hydroxycitric acid**. Controlled release can be used to provide a sustained and modulated amount of the active to the body as desired and therefore regulate the use of the compound as a hypotensive agent.

L78 ANSWER 42 OF 47 USPATFULL

ACCESSION NUMBER:	2001:59921 USPATFULL			
TITLE:	Magnesium (-)hydroxycitrate, method of preparation, applications, and compositions in particular pharmaceutical containing same			
INVENTOR(S):	Shrivastava, Ravi, 43bis route de Chateaugay, 63118 Cebazat, France Lambropoulos, Patrick, 35 Traverse Nicolas, 13007 Marseille, France			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6221901	B1	20010424	
	WO 9817671		19980430	
APPLICATION INFO.:	US 1999-284864		19990422	(9)
	WO 1997-FR1860		19971017	
			19990422	PCT 371 date
			19990422	PCT 102(e) date
	NUMBER		DATE	
PRIORITY INFORMATION:	FR 1996-13094		19961022	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	O'Sullivan, Peter			
LEGAL REPRESENTATIVE:	Browdy and Neimark			
NUMBER OF CLAIMS:	23			
EXEMPLARY CLAIM:	1			
LINE COUNT:	508			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Magnesium (-)hydroxycitrate, preparation process, dietary and therapeutic uses particularly in the cardiovascular field, and compositions in particular pharmaceutical containing it.

L78 ANSWER 43 OF 47 EUROPATFULL COPYRIGHT 2002 WILA

GRANTED PATENT - ERTEILTES PATENT - BREVET DELIVRE

ACCESSION NUMBER:	810868 EUROPATFULL EW 200135 FS PS
TITLE:	USE OF PIPERINE AS A GASTROINTESTINAL ABSORPTION ENHANCER. VERWENDUNG VON PIPERINE ZUR VERBESSERUNG DER GASTROINTESTINALEN ABSORPTION. UTILISATION DE LA PIPERINE POUR AUGMENTER L'ABSORPTION GASTROINTESTINALE.
INVENTOR(S):	MAJEED, Muhammed, Unit 6 121 Ethel Road West, Piscataway, NJ 08854, US; BADMAEV, Vladimir, Unit 6 121 Ethel Road West, Piscataway, NJ 08854, US; RAJENDRAN, R., Jayanagar Eastend, 1382 Southend Main Road, 9th Block, Bangalore 560 069, IN
PATENT ASSIGNEE(S):	Sabinsa Corporation, Unit 6, 121 Ethel Road West, Piscataway, NJ 08854, US
PATENT ASSIGNEE NO:	2199040
AGENT:	Huber, Bernhard, Dipl.-Chem. et al., Weickmann & Weickmann Patentanwaelte Kopernikusstrasse 9, 81679

Muenchen, DE
AGENT NUMBER: 5835
OTHER SOURCE: BEPB2001037 EP 0810868 B1 0020
SOURCE: Wila-EPS-2001-H35-T1
DOCUMENT TYPE: Patent
LANGUAGE: Anmeldung in Englisch; Veroeffentlichung in Englisch
DESIGNATED STATES: R AT; R BE; R CH; R DE; R DK; R ES; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R MC; R NL; R PT; R SE
PATENT INFO.PUB.TYPE: EPB1 EUROPAEISCHE PATENTSCHRIFT (Internationale Anmeldung)
PATENT INFORMATION:

PATENT NO	KIND	DATE
EP 810868	B1	20010829
'OFFENLEGUNGS' DATE:		19971210
APPLICATION INFO.: EP 1995-939489		19951106
PRIORITY APPLN. INFO.: US 1995-393738		19950224
US 1995-550496		19951030
RELATED DOC. INFO.: WO 95-US12758		951106 INTAKZ
WO 9625939		960829 INTPNR
REFERENCE PAT. INFO.: US 4284657 A		

REF. NON-PATENT-LIT.: C. K. ATAL ET AL.: "SCIENTIFIC EVIDENCE ON THE ROLE OF AYURVEDIC HERBALS ON BIOAVAILABILITY OF DRUGS" JOURNAL OF ETHNOPHARMACOLOGY, vol. 4, no. 2, September 1981, pages 229-232, XP002077891 R. K. JOHRI ET AL.: "AN AYURVEDIC FORMULATION 'TRIKATU' AND ITS CONSTITUENTS." JOURNAL OF ETHNOPHARMACOLOGY, vol. 37, no. 2, September 1992, pages 85-91, XP002077892 C. K. MATHAI: "A MODIFIED EXTRACTION AND ESTIMATION METHOD OF OLEORESIN AND PIPERINE IN BLACK PEPPER /PIPER NIGRUM L.) BERRIES." INDIAN SPICES, vol. 25, no. 2/3, 1988, pages 3-5, XP002077893 CHEMICAL ABSTRACTS, No. 110:6454, WOOD et al., "Piperine Determination in Pepper and Its Oleoresins-a Reversed-Phase High Performance Liquid Chromatographic Method"; & FLAVOUR FRAGRANCE JOURNAL, Vol. 3(2), issued 1988, 55-64

L78 ANSWER 44 OF 47 EUROPATFULL COPYRIGHT 2002 WILA

GRANTED PATENT - ERTEILTES PATENT - BREVET DELIVRE

ACCESSION NUMBER: 414730 EUROPATFULL EW 199950 FS PS
TITLE: CHEMICAL COMPOUNDS AND PHARMACEUTICAL COMPOSITIONS CAPABLE OF RELEASING A DRUG.
CHEMISCHE VERBINDUNGEN UND PHARMAZEUTISCHE ZUSAMMENSETZUNGEN ZUR FREISETZUNG VON ARZNEIMITTELN.
COMPOSES CHIMIQUES ET COMPOSITIONS PHARMACEUTIQUES CAPABLES DE DELIVRER UN MEDICAMENT.
INVENTOR(S): Mills, Randell L., R.D. 2, Cochranville Pennsylvania 19330, US
PATENT ASSIGNEE(S): Mills, Randell L., R.D. 2, Cochranville Pennsylvania 19330, US
PATENT ASSIGNEE NO: 745290
AGENT: Beetz & Partner Patentanwaelte, Steinsdorfstrasse 10, 80538 Muenchen, DE
AGENT NUMBER: 100712
OTHER SOURCE: EPB1999067 EP 0414730 B1 991215
SOURCE: Wila-EPS-1999-H50-T1
DOCUMENT TYPE: Patent
LANGUAGE: Anmeldung in Englisch; Veroeffentlichung in Englisch
DESIGNATED STATES: R AT; R BE; R CH; R DE; R FR; R GB; R IT; R LI; R LU; R NL; R SE
PATENT INFO.PUB.TYPE: EPB1 EUROPAEISCHE PATENTSCHRIFT (Internationale

Anmeldung)

PATENT INFORMATION:

PATENT NO	KIND	DATE
EP 414730	B1	19991215
'OFFENLEGUNGS' DATE:		19910306
APPLICATION INFO.: EP 1989-904951		19890331
PRIORITY APPLN. INFO.: US 1988-175970		19880331
RELATED DOC. INFO.: WO 89-US1361		890331 INTAKZ
WO 8909833		891019 INTPNR
REFERENCE PAT. INFO.: WO 83-04255 A		US 3798131 A
US 4599303 A		US 4626501 A
US 4656127 A		US 4683194 A
US 4716106 A		

REF. NON-PATENT-LIT.: STN FILE SERVER & FILE CA (KARLSRUHE) & CHEMICAL ABSTRACTS, vol. 94, no. 9, 1980, Columbus, Ohio, US; abstract no. 63484, ANNA D. INGLOT 'DEXTRAN T FRACTIONS SUBSTITUTED WITH CIBACRON BLUE F3G-A AS CARRIERS FOR MOUSE INTERFERON'

L78 ANSWER 45 OF 47 EUROPATFULL COPYRIGHT 2002 WILA

PATENT APPLICATION - PATENTANMELDUNG - DEMANDE DE BREVET

ACCESSION NUMBER: 815857 EUROPATFULL EW 199802 FS OS

TITLE: ANTIOBESTIC AGENT CONTAINING PROCYANIDIN AS THE ACTIVE INGREDIENT.
PROCYANIDIN ALS DEN AKTIVEN BESTANDTEIL ENTHALTENDE MITTEL GEGEN FETTLEIBIGKEIT.
AGENT ANTI-OBESITE DONT LE PRINCIPE ACTIF EST LA PROCYANIDINE.

INVENTOR(S): NAKAHARA, Koichi, 3-4-A211, Higashiizumigaoka Toyonaka-shi, Osaka 560, JP;
NAKAI, Masaaki, 6-17-5-B103, Senriyamanishi Suita-shi, Osaka 565, JP;
TAMURA, Yukiyoshi, 1348-7, Mukaishimamachi Mitsugi-gun, Hiroshima 722, JP

PATENT ASSIGNEE(S): SUNTORY LIMITED, 1-40, Dojimahama 2-chome, Kita-ku, Osaka-shi, Osaka 530, JP

PATENT ASSIGNEE NO: 423903

AGENT: Hansen, Bernd, Dr. Dipl.-Chem. et al, Hoffmann Eitle, Patent- und Rechtsanwaelte, Arabellastrasse 4, 81925 Muenchen, DE

AGENT NUMBER: 4924

OTHER SOURCE: ESP1998001 EP 0815857 A1 980107

SOURCE: Wila-EPZ-1998-H02-T1b

DOCUMENT TYPE: Patent

LANGUAGE: Anmeldung in Japanisch; Veroeffentlichung in Englisch; Verfahren in Englisch

DESIGNATED STATES: R AT; R BE; R CH; R DE; R DK; R ES; R FI; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R NL; R PT; R SE

PATENT INFO.PUB.TYPE: EPA1 EUROPAEISCHE PATENTANMELDUNG (Internationale Anmeldung)

PATENT INFORMATION:

PATENT NO	KIND	DATE
EP 815857	A1	19980107
'OFFENLEGUNGS' DATE:		19980107
APPLICATION INFO.: EP 1996-943298		19961226
PRIORITY APPLN. INFO.: JP 1995-338493		19951226
RELATED DOC. INFO.: WO 96-JP3810		961226 INTAKZ
WO 9723210		970703 INTPNR

ABEN An antiobestic agent which has, in addition to the antiobestic effect,

the effects of inhibiting saccharolytic digestive enzymes, suppressing an increase in blood sugar level, inhibiting the absorption of monosaccharides, adsorbing and excreting cholic acid, lowering cholesterol level and blood triglyceride level and, inhibiting lipase and is useful not only as an antiobestic agent but also as antilipotrophic, antihyperlipidemic, antiarteriosclerotic and antidiabetic agents. An extract of tamarind seed coat being rich un procyanidin (trimer of formula (I)), which is the active ingredient in the antiobestic agent, exerts as such a potent antiobestic effect without being purified any more. The antiobestic agent serves as a saccharolytic digestive enzyme inhibitor, a hypoglycemic agent, a monosaccharide absorption inhibitor, a cholic acid adsorption/excretion agent, a cholesterol-lowering agent, a blood triglyceride level-lowering agent and a lipase inhibitor and facilitates the production of foods, drinks and feeds showing these effects, thus contributing to the amelioration or prevention of diabetics or obesity in daily life.

L78 ANSWER 46 OF 47 EUROPATFULL COPYRIGHT 2002 WILA

PATENT APPLICATION - PATENTANMELDUNG - DEMANDE DE BREVET

ACCESSION NUMBER: 803202 EUROPATFULL EW 199744 FS OS
TITLE: Dietary composition containing chitosan, Garcinia cambogia hydroxycitrate and organic chromium.
Chitosan, Garcinia cambogia/-hydroxycitrat und organisches Chrom enthaltende Diaetzusammensetzung.
Composition dietetique comprenant chitosane, hydroxycitrate de Garcinia cambogia et chrome organique.
INVENTOR(S): Littera, Renato, Via Barbaro, 19, 10143 Torino, IT
PATENT ASSIGNEE(S): SIRC S.p.A. NATURAL & DIETETIC FOODS, Via E. Fermi, 3, I-20090 Caleppio Di Settala (MI), IT
PATENT ASSIGNEE NO: 1617230
AGENT: Sarpi, Maurizio, Studio FERRARIO Via Collina, 36, 00187 Roma, IT
AGENT NUMBER: 41002
OTHER SOURCE: ESP1997065 EP 0803202 A2 971029
SOURCE: Wila-EPZ-1997-H44-T3a
DOCUMENT TYPE: Patent
LANGUAGE: Anmeldung in Italienisch; Veroeffentlichung in Englisch; Verfahren in Englisch
DESIGNATED STATES: R AT; R BE; R CH; R DE; R DK; R ES; R FI; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R MC; R NL; R PT; R SE
PATENT INFO.PUB.TYPE: EPA2 EUROPAEISCHE PATENTANMELDUNG
PATENT INFORMATION:

PATENT NO	KIND	DATE
EP 803202	A2	19971029

'OFFENLEGUNGS' DATE: 19971029
APPLICATION INFO.: EP 1997-830189 19970424
PRIORITY APPLN. INFO.: IT 1996-RM96279 19960426

ABEN The use of preparations based on the combination of chitosan with organic chromium and Garcinia cambogia **hydroxycitrate** as dietary products for the treatment of obesity having hypocholesteremic and sugar absorption reducing activity is disclosed.

The proposed combination of chitosan with organic chromium and Garcinia cambogia **hydroxycitrate** is formulated on the base of the effects that the above three components have on the glucid metabolism. Such effects tends particularly to decrease the values of cholesterolemia and triglycerides in case they are too high.

The integrator of the invention can be administered by mouth in the usual dose unit both as capsules and tablets and is efficacious as diet integrator in the weight reducing programs aiming at calorie

restrictions in obese subjects, in the treatment of **hypertension** , and as hypocholesteremic product.

L78 ANSWER 47 OF 47 EUROPATFULL COPYRIGHT 2002 WILA

GRANTED PATENT - ERTEILTES PATENT - BREVET DELIVRE

ACCESSION NUMBER: 669832 EUROPATFULL EW 199639 FS PS
TITLE: METHOD FOR TREATMENT OR PREVENTION OF OBESITY.
METHODE ZUR BEHANDLUNG ODER VERHUeTUNG VON FETTLEIBIGKEIT.
PROCEDE DE TRAITEMENT OU DE PREVENTION DE L'OBESITE.
INVENTOR(S): CLARK, Ross G., 711 Ursula Avenue, Pacifica, CA 94044, US
PATENT ASSIGNEE(S): GENENTECH, INC., 460 Point San Bruno Boulevard, South San Francisco, CA 94080-4990, US
PATENT ASSIGNEE NO: 210485
AGENT: Ellis, Edward Lovell et al, MEWBURN ELLIS York House 23 Kingsway, London WC2B 6HP, GB
AGENT NUMBER: 30421
OTHER SOURCE: EPB1996062 EP 0669832 B1 960925
SOURCE: Wila-EPS-1996-H39-T1
DOCUMENT TYPE: Patent
LANGUAGE: Anmeldung in Englisch; Veroeffentlichung in Englisch
DESIGNATED STATES: R AT; R BE; R CH; R DE; R DK; R ES; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R MC; R NL; R PT; R SE
PATENT INFO.PUB.TYPE: EPB1 EUROPAEISCHE PATENTSCHRIFT (Internationale Anmeldung)
PATENT INFORMATION:

PATENT NO	KIND	DATE
EP 669832	B1	19960925
'OFFENLEGUNGS' DATE:		19950906
APPLICATION INFO.:		19931026
PRIORITY APPLN. INFO.:	US 1992-968623	19921029
RELATED DOC. INFO.:	WO 93-US10259	931026 INTAKZ
	WO 9409813	940511 INTPNR
REFERENCE PAT. INFO.:	EP 331630 A	EP 473084 A
	WO 91-18621 A	WO 92-13556 A

REF. NON-PATENT-LIT.: FASEB JOURNAL vol. 6, no. 5, 28 February 1992, BETHESDA, MD US page A1676 D.B. HAUSMAN ET AL. 'EFFECT OF SOMATOTROPIN TREATMENT ON ADIPOSE CELL METABOLISM IN OBESE ZUCKER RATS WITH RESTRICTED CALORIC INTAKE.'

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FILE 'NAPRALERT' ENTERED AT 15:23:34 ON 18 JAN 2002
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FILE COVERS 1650 TO 14 JAN 2002 (20020114/ED)

This file contains CAS Registry Numbers for easy and accurate
substance identification.


L2             5 SEA FILE=REGISTRY ABB=ON  27750-10-3 OR 64913-19-5 OR 132436-67
                 -0 OR 185196-38-7 OR 213385-58-1
L57           31 SEA FILE=NAPRALERT ABB=ON  L2 OR HYDROXYCITRATE OR (HIBISCUS
                 OR GARCINIA OR HYDROXYCITRIC OR HYDROXY CITRIC OR HYDROCITRIC)(
                 W)ACID
L58         1279 SEA FILE=NAPRALERT ABB=ON  HYPERTENS? OR ANTIHYPERTENS?
L59          549 SEA FILE=NAPRALERT ABB=ON  BLOOD PRESSURE
L60          535 SEA FILE=NAPRALERT ABB=ON  HYPERINSULIN? OR INSULIN?
L61           43 SEA FILE=NAPRALERT ABB=ON  GLUCOCORTICOID# OR HYDROXYCORTICOSTE
                 ROID# OR (GLUCO CORTICOID#) OR (HYDROXYCORTICO OR HYDROXY
                 CORTICO)(W)STEROID#
L62            1 SEA FILE=NAPRALERT ABB=ON  L57 AND (L58 OR L59 OR L60 OR L61)

=> d qrd 162

L62  ANSWER 1 OF 1 NAPRALERT   COPYRIGHT (C) 2002 BD. TRUSTEES, U. IL.
AN   95:3335  NAPRALERT
DN   K19506
TI   HEXOSE METABOLISM IN PANCREATIC ISLETS. EFFECT OF (-)-
     HYDROXYCITRATE UPON FATTY ACID SYNTHESIS AND INSULIN
     RELEASE IN GLUCOSE-STIMULATED ISLETS
AU   SENER A; MALAISSE W J
CS   LAB EXP MED, BRUSSELS FREE UNIV, BRUSSELS B-1000 BELGIUM
SO   BIOCHIMIE (1991) 73 (10) p. 1287-1290.
DT   Journal
LA   ENGLISH
OS   CA 116:76791
CHC  844
ORGN Class: DICOT
      TYPE OF STUDY (STY): IN VITRO.  Classification (CC): CITRATE LYASE
          INHIBITION
          Dosage Information: RAT; CONC USED: 5.0 MILLIMOLS
          Pathological system: ISLETS OF LANGERHAN
          Qualitative results: ACTIVE
          Comment(s): DATA INCOMPLETE - DERIVED FROM AN ABSTRACT.
                      CMPD DID NOT AFFECT GLUCOSE-STIMULATED INSULIN
                      RELEASE OR INCORPORATION OF LABELED ACETATE INTO LIPIDS.
          COMPOUND. Chemical name (CN): CITRIC ACID,HYDROXY: (-)
               CAS Registry Number (RN): 27750-10-3
               Class identifier (CI): MISCELLANEOUS
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FILE 'HOME' ENTERED AT 15:23:52 ON 18 JAN 2002
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